Childhood obesity has now reached epidemic proportions, and large numbers of children are presenting for medical care. The Royal College of Paediatrics and Child Health (RCPCH) has produced a document contending that most of these children should be seen in primary care. However the House of Commons Health Select Committee sees otherwise and has called upon the National Health Service to ensure obese children have access to specialist care.

The paediatrician’s role in managing obesity therefore needs clarifying, particularly in the context that any recommendations for identification, investigation, and treatment have enormous financial and service implications.

In this article I map out what is known about the diagnosis, epidemiology, complications, and treatment of the condition, and conclude by examining published guidelines with some thoughts about the shape that services might take.

THE DIAGNOSIS OF OBESITY

Obesity can simply be defined as an excess of body fat. Although there are more accurate methods for measuring body fat, body mass index (BMI) is the only method that is feasible outside the realms of research. BMI is calculated from the formula BMI = weight (kg)/height² (m).

By convention in the UK, the cut-offs for overweight and obesity are taken to be the 91st and 98th centiles on the 1990 UK reference charts. These cut-offs have some rationale as epidemiological studies show that the risk for eventual morbidity significantly increases above the 95th centile. There are two versions of the BMI charts (fig 1). The version for managing obesity is recommended (fig 1A rather than 1B) as it provides extra centile lines above the 99.6th to allow for mapping the progress of severely obese children over time.

EPIDEMIOLOGY

Childhood obesity has shown a dramatic increase in the last decade. Figures vary as there is much inconsistency in how obesity is defined. Our data in Leeds showed that as many as 30% of 11 year olds were overweight (> 85th centile) and 17% were obese (>95th centile) with a significant rise over only a three year period.

The problem of epidemiological definition remains under debate. In adults the criteria for obesity and overweight is taken as BMI ≥ 30 kg/m² and ≥ 25 kg/m², respectively, and these have been derived from analysis of health risk. We do not have the same robust evidence for deciding on cut-offs for children.

The International Obesity Task Force (IOTF) recently recommended that we use an extrapolation of the adult cut-offs back through to childhood. Using data from six countries BMI cut-offs for each age group have been calculated to allow for international comparison. These IOTF criteria are now marked on the new UK charts (fig 1). They are principally of value epidemiologically and it is important to note that the IOTF criteria are more stringent than the cut-offs used clinically.

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withstand scarcity by storing fat. In this era of plenty, where we are also relatively inactive, children are laying down adipose tissue at an alarming rate with dire consequences.

DIFFERENTIAL DIAGNOSIS
When evaluating a child for obesity, it is important to consider causes other than nutritional obesity. These are shown in table 1, but are rare. They can generally be suspected on clinical examination.

The growth chart provides the first clue to a non-nutritional cause as illustrated in fig 2. In nutritional obesity (fig 2B) the over nutrition tends to promote growth, and tall stature therefore suggests this diagnosis. It is worth noting that tall stature is not necessarily sustained through to adult height, as puberty tends to occur early. Short stature with obesity suggests a syndromic or hypothalamic cause (fig 2A), of which the most common are Prader Willi and Bardet Biedl syndrome. Slowing in growth velocity in conjunction with weight gain indicates an endocrine cause (fig 2C), such as hypothyroidism, hypercortisolism, or growth hormone deficiency. Endocrine causes are vital to identify as they are treatable.

Clinical features that suggest that the obesity may be a result of a genetic syndrome are shown in table 2.

Table 1 Causes of obesity

<table>
<thead>
<tr>
<th>Category</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Common causes</td>
<td>Nutritional (exogenous obesity)</td>
</tr>
<tr>
<td></td>
<td>Psychosocial</td>
</tr>
<tr>
<td>Rare causes</td>
<td>Syndromes— for example, Prader Willi, Bardet-Biedl</td>
</tr>
<tr>
<td>Endocrine</td>
<td>Hypothyroidism</td>
</tr>
<tr>
<td></td>
<td>Cushings</td>
</tr>
<tr>
<td></td>
<td>Hypothalamic lesions</td>
</tr>
<tr>
<td></td>
<td>Single gene defects</td>
</tr>
</tbody>
</table>

*Listed separately from nutritional to ensure that psychosocial causes are not missed as a primary issue.

CONSEQUENCES OF OBESITY
Table 3 shows the complications that occur as a consequence of obesity. Undoubtedly the most common problems are psychosocial, and it is likely that most obese children seeking medical referral are suffering from bullying (either victim or aggressor), poor self esteem, or social problems. Other morbidities include orthopaedic problems such as slipped capital femoral epiphyses, Blount’s disease, and leg and back pain. Polycystic ovary syndrome presents with menstrual irregularities (usually amenorrhoea), hirsutism, and acne. Obstructive sleep apnoea is a further concern and presents with disturbed sleep, snoring, and lethargy during the day.

Until recently the medical consequences seemed to be rare, and childhood obesity was essentially considered to be a cosmetic problem. However, the evidence is now emerging that a majority of obese children already have indicators of organic disease. A study in the USA reported that 58% of children with a BMI above the 95th centile have hypertension, hyperlipidaemia, or insulin resistance, and 25% have two or more of these.4

A major issue that we need to address, in the light of this evidence, is how actively we should be looking for evidence of the metabolic syndrome (hypertension, dyslipidaemia, and impaired glucose tolerance) and fatty liver disease. This is recommended in a recent International Consensus Statement7 but requires some debate in this country, in view of the significant cost implications involved.

MANAGEMENT OF OBESITY
What should the paediatrician’s role be in managing obesity?
Paediatricians often find the management of obesity to be unrewarding, and probably would be quite content if it was considered to be the remit of primary care. The principal problem is that most find it hard to help children achieve a meaningful reduction in weight through periodic attendance at a general or paediatric endocrinology clinic.

It is therefore helpful to clarify the goals of paediatric care as:

1. Healthy lifestyle
2. Weight loss
3. Psychosocial support
4. Medical monitoring
5. Prevention of complications
Identification of a medical cause
Identification of any consequences of obesity
Promotion of weight loss/control
Management of medical problems.

Diagnosis of the cause and consequences of obesity sit comfortably in the paediatric setting. However, if one is to go beyond diagnosis and attempt to intervene, the critical ingredients are good communication skills and adequate time to focus on lifestyle issues and how change can be achieved.

The diagnostic process

Table 4 summarises the essential features of the clinical evaluation. Weight and height need to be plotted on a growth chart. It is helpful to use a BMI chart as well, as this allows a more accurate way of assessing weight gain in relation to gain in height.

Waist circumference is another measure worthy of mention (fig 3). It is theoretically of importance as it relates to cardiovascular morbidity in adults. However, in practice it is difficult to attain consistently. Three different methods of measurement have been recommended: midway between bony markers; at the skin crease on lateral flexion; or in relation to the umbilicus. These are all hard to obtain in an obese child—bony landmarks are hard to feel, there is often more than one lateral crease, and the abdomen may be so pendulous that the umbilicus is an uncertain marker. More work is therefore needed before waist circumference can be considered a useful measure.

Table 2: Clinical features suggesting a genetic syndrome associated with obesity
- Short stature
- Severe unremitting obesity
- Onset of obesity before the age of 2 years
- Dysmorphic features
- Microcephaly
- Learning disability
- Hypotonia
- Hypogonadism
- Eye abnormalities
- Skeletal abnormalities
- Sensorineural deafness
- Renal abnormalities
- Cardiac abnormalities

Table 3: Consequences for the obese child
Overt
- Emotional and behavioural
- Orthopaedic
  - Blount’s disease
  - Slipped capital femoral epiphyses
- Asthma
- Sleep apnoea
- Pseudotumour cerebri
- Polycystic ovary syndrome
Occult
- Impaired glucose tolerance
- Hypertension
- Dyslipidaemia
- Steatohepatitis

Table 4: Clinical evaluation of the obese child
<table>
<thead>
<tr>
<th>History</th>
<th>Physical examination</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age of onset</td>
<td>Measuring and plotting growth: height, weight, BMI, waist circumference</td>
</tr>
<tr>
<td>Diet and eating behaviour</td>
<td>Blood pressure</td>
</tr>
<tr>
<td>Physical activity and inactivity</td>
<td>Signs of Cushing’s/hypothyroidism</td>
</tr>
<tr>
<td>School and social issues</td>
<td>Acanthosis nigricans</td>
</tr>
<tr>
<td>Sleep problems and daytime somnolence</td>
<td>Hirsutism</td>
</tr>
<tr>
<td>Hip/knee pain</td>
<td></td>
</tr>
<tr>
<td>Menstrual irregularities</td>
<td></td>
</tr>
<tr>
<td>Symptoms of hypothyroidism</td>
<td></td>
</tr>
<tr>
<td>Family history of obesity</td>
<td></td>
</tr>
<tr>
<td>Family history of early heart disease</td>
<td></td>
</tr>
</tbody>
</table>
Table 5 shows investigations that can be considered in obesity. If on clinical evaluation it is clear that the child has nutritional obesity there is no real need to carry out endocrine or genetic investigations, although without results in hand some families may remain convinced that there is a “glandular problem”. Hypothyroidism and Cushing’s very rarely present with obesity, but if suspected, investigations are required. If features are present that suggest a genetic syndrome (table 2) genetic investigations are required.

It is debatable whether investigation for morbidity is required. Children seen in paediatric endocrinology clinics are likely to be investigated whereas those in community or primary care are not (although there is no evidence that these populations differ in terms of degree of obesity). One can argue that as management for all these morbidities in the first instance is weight loss, there is little to be gained by carrying out laboratory investigations to ascertain their presence. However, it is hard to argue that ignorance should be a policy and the prevalence of morbidity does indicate that the yield from tests will be high. A major consideration is whether knowledge of co-morbidity will benefit and motivate the family (or perhaps the opposite).

**What are we hoping to achieve?**

Ideally one would hope that effective management would aim to return children to a BMI where morbidities are reduced (that is, below the 95th centile). Certainly that would concur with our patients’ desires, but is clearly an unrealistic goal. Classic paediatric dogma states that we should be aiming for maintaining weight while allowing the child to grow and so reduce their BMI. This approach may be valid for the early years of childhood, but is hardly appropriate for a teenager at 170 kg who has little growing left.

The evidence from the literature provides some guidance regarding a realistic goal. Systematic reviews indicate that with aggressive treatment, reduction in overweight by 10–20% may be possible in the short term at least. The adult literature informs us that a weight reduction of 10% brings health benefits. Perhaps a reduction in BMI over time of 10% should therefore be set as a target.

**What interventions are effective?**

The evidence base for treatment of such a common and important condition of childhood is sadly minimal. Systematic reviews that focus on lifestyle interventions in children emphasise that the evidence from trials is very limited. They describe 18 trials of adequate quality to be included. All are small, with 11 having a total sample size of less than 50 participants. Some short term success has been achieved by interventions involving a combination of dietary advice, behavioural modification, and physical activity. However the reviewers emphasise that these trials were in the most part carried out in academic tertiary care centres with highly specialised staff involving white, middle class, motivated families. Their generalisability to the paediatrician working solo in outpatients is necessarily limited. Only two treatment trials have been carried out in Europe, and none in the UK, so it is

![Figure 3 Waist circumference charts for (A) boys and (B) girls](http://ep.bmj.com/)

<table>
<thead>
<tr>
<th>Table 5</th>
<th>Investigations in the obese child</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Causes</strong></td>
<td><strong>Consequences</strong></td>
</tr>
<tr>
<td>Thyroid function tests</td>
<td>Fasting lipid screen</td>
</tr>
<tr>
<td>Urinary free cortisol or diurnal cortisol</td>
<td>Liver function tests</td>
</tr>
<tr>
<td>Genetic studies</td>
<td>Urine glucose</td>
</tr>
<tr>
<td>Chromosomes</td>
<td>Fasting glucose and insulin</td>
</tr>
<tr>
<td>DNA for Prader-Willi</td>
<td>Oral glucose tolerance test</td>
</tr>
<tr>
<td>GOOS</td>
<td>If at high risk for diabetes</td>
</tr>
<tr>
<td>DNA for Prader-Willi</td>
<td>Sex hormone binding globulin, FSH, LH for PCOS</td>
</tr>
<tr>
<td></td>
<td>O2 monitoring for sleep apnoea</td>
</tr>
</tbody>
</table>

*Genetics of Obesity Study in Cambridge.*

FSH, follicle stimulating hormone; LH, luteinising hormone; PCOS, polycystic ovary syndrome.
particularly difficult to translate the results into useful information for developing services in this country.

The reviews did not address drug or surgical treatments of paediatric obesity, although there are a few small scale trials now underway in the USA.7

**Lifestyle change**

The mainstay of management for obese children is lifestyle change. However, our conventional approach seems to be singularly ineffective and is the reason why the majority of paediatricians find working in the field to be so unrewarding. Patient opinion informs us that families do not respond well to medical prophets of doom, and report that the traditional dietetic approach is also unhelpful.

There is little to guide us as to the way forward. For most families, knowledge of what is required is not the issue. The major difficulty is in encouraging major behavioural change. In order to do this, time is of the essence; without frequent contact for encouragement and support, success is unlikely.

The current dietetic approach is to work towards a healthy balanced diet with reduction in high energy foods. Low fat and low carbohydrate trials in adolescence are underway in the USA7 but none are currently recommended in childhood. While these diets can induce short term success, they are likely to precipitate a binge–diet cycle that is more than unproductive.

Physical activity may be a more achievable goal than major dietary change. Some obese children enjoy being active but often withdraw from engaging publicly because of fear of ridicule. There is also some evidence that reduction in sedentary behaviours (such as television viewing) may be more effective than promoting exercise. These aspects of lifestyle change are too often neglected by health professionals.

Hospital specialty clinics are not likely to be the location where lifestyle change can be effectively induced. Community, school, and even social services may need to be engaged for the severely obese child, and liaising with these services may be particularly helpful for the child with learning disability.

**Drug treatments**

It is important to emphasise that lifestyle change must be the primary approach for the obese child and medication should only ever be considered in combination with a diet and exercise programme. So saying, no drugs for the treatment of obesity are licensed for the paediatric age group in this country. The following are currently used in adult obesity:

- **Orlistat** acts by inhibiting pancreatic lipase and increasing faecal losses of triglyceride
- **Sibutramine** induces anorexia by inhibiting neuronal reuptake of serotonin and noradrenaline (norepinephrine)
- **Metformin** is an antidiabetic drug which induces weight loss in some individuals.

Orlistat is occasionally prescribed in endocrinology clinics but it is generally poorly tolerated as it induces flatulence and diarrhoea. Sibutramine is licensed for use in adolescents in the USA and has undergone a small trial with some success.7 However there are potentially serious side effects which obviate its use at this time. Metformin offers some promise as it causes decreased food intake and weight loss in a proportion of adults. Two small trials in the USA suggest modest benefits for the child with insulin resistance,7 and a further multicentre trial is now being proposed in the UK to be run under the auspices of the RCPCH. Lastly octreotide, a somatostatin analogue, is undergoing trials in the USA for children with hypothalamic obesity.

**Surgery**

As the long term success of lifestyle intervention and pharmacotherapy in severe obesity has been so disappointing, more aggressive approaches such as surgery are already being considered in extreme obesity where severe co-morbidity is already present. The two approaches are the laparoscopic banding procedure and Roux-en-Y gastric bypass. Studies of efficacy and safety are now underway in the USA,7 and may well be the only solution for the morbidly obese. Clearly these cannot be considered except in specialist centres with a designated paediatric multidisciplinary team.

**CLINICAL GUIDELINES FOR THE TREATMENT OF OBESITY**

In April 2003 the Scottish Intercollegiate Guidelines Network produced its guidelines on the management of obesity in children and young people,11 and these were subsequently appraised by the RCPCH13 (table 6). The guidelines are a disappointment to the paediatrician seeking the best way to provide help for their obese patients. They have been rigorously compiled, but cannot offer much guidance due to the lack of quality evidence, particularly in the area of treatment. They are unsubstantiated by any grade A or B level

### Table 6: Scottish Intercollegiate Guidelines Network: management of obesity in children and young people

| BMI centile should be used to identify childhood obesity | C | C |
| Obsese children have a BMI >98th centile | D | No comment |
| Treatment should only be considered where | D |
| – A child is defined as obese | D |
| – And the family are ready to make the necessary lifestyle changes | D |
| In most obese children weight maintenance is an acceptable goal | D |
| Weight maintenance and/or weight loss can be achieved by sustained behavioural changes—for example, healthier eating, increased physical activity, reducing physical inactivity | D |
| Prevention and treatment of obesity should be initiated in childhood | C |
| Parental obesity should be recognised as a risk factor for childhood obesity to persist into adulthood | C |
| The following should be referred to a paediatric consultant before treatment is considered: | C |
| – Children who may have a serious obesity related morbidity | C |
| – Children with a suspected underlying medical cause (including all <24 months of age) | C |
| – All children with BMI >99.6th centile | C |
| School family and societal interventions should be considered for the prevention of obesity in children | C |

C, evidence from well conducted case control or cohort studies; D, evidence from non-analytic studies or expert opinion.

RCPCH, Royal College of Paediatrics and Child Health; SIGN, Scottish Intercollegiate Guidelines Network.
recommendations and rely entirely on expert opinion regarding treatment. They also offer very little detail regarding the position of secondary care (table 7), which is where most paediatricians have a role.

**WHAT SHAPE SHOULD SERVICES TAKE?**

It is hard to come up with recommendations for the shape of services at primary and secondary level in the absence of clear evidence for effective treatment. It would also be irresponsible at this time to map out a plan for expensive clinical services. On the other hand, unless we focus on the problem and begin to allocate resources, we shall never develop the body of expertise required to tackle it. There is already an increasing demand from the public for clinical care, and in my view it is inappropriate to consign the burden of the service to primary care where obese (but apparently healthy) children cannot hope to receive adequate attention from overstretched staff.

We also cannot wait too long for quality evidence to emerge. Clinical trials are expensive and take time to run. In order to move the agenda forward we should be promoting research in this area, but also allocating resources to secondary care to set a basis for what is required. Key staff must include paediatric, nursing, mental health, dietetic, and exercise professionals, although frontline care may well not involve all of these individuals. Whatever evidence eventually emerges, it is likely that children will need encouragement, understanding, and support, and will need to be seen on a very frequent basis.

**Who at this time should be seen in a specialist clinic?**

Obesity services certainly suffer from postcode allotment. Children are referred for specialist attention somewhat randomly, and as screening has been discounted for the time being, this situation is likely to continue. It is reasonable to suggest that dedicated clinics are required to see the following:

- Children who may have a medical cause for their obesity
- Children with existing co-morbidity (when this emerges) or a strong family history of diabetes and early heart disease
- Children with significant emotional and behavioural problems related to obesity
- Children with severe obesity. We have no evidence based definition to use at this time but perhaps might arbitrarily take a BMI of > 3.5 SD, which is roughly equivalent to the adult definition of morbidity obesity (BMI > 40).

**FUTURE WORK/RESEARCH REQUIRED: EVIDENCE TO FORMULATE BEST PRACTICE**

It is hard to write a best practice article with so little evidence on which to base it. However, it is clear what is required to move the agenda forward. This includes the following:

1. Quality randomised controls of lifestyle interventions in non-specialist settings
2. A randomised controlled trial of metformin and perhaps other medications now used in adult obesity
3. Experience and rigorous evaluation of surgical procedures for the exceptionally obese adolescent in specialised centres
4. Public health monitoring of the epidemic
5. Epidemiological studies to determine how the severity of obesity and other risk factors relate to subsequent morbidity and mortality.

**SUMMARY**

- We are in the midst of an epidemic of obesity in which we have no effective treatment strategies
- Specialist clinics are required to meet demand and take the agenda forward, although this may be difficult to achieve given the lack of evidence for treatment
- Paediatricians have a clear role in identifying the rare cases where there is a medical cause, and in managing frank co-morbidity
- Issues are less clear when it comes to identifying co-morbidities (which are extremely prevalent)
- The rate limiting step in mapping out best practice is the lack of effective treatment
- There is no justification for introducing screening until we have effective treatment, and more harm than good may be achieved by doing so.

**REFERENCES**

13. Genetics of Obesity Study. University of Cambridge. Details from Dr S Farooq, Addenbrooke’s Hospital, Cambridge CB2 2QR.

**ADDITIONAL RECOMMENDED READING**


The obese child

Mary C J Rudolf

Arch Dis Child Educ Pract Ed 2004 89: ep57-ep62
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